EDITORIAL

ORIGINAL ARTICLES
• Osteoporosis and bone health in Diabetes
• The Chain of DKA Management - what can go wrong and how do you fix it?
• Insulin Resistance... the Psychological Kind. Emotional Aspects of Injecting Insulin
• Use of additional technologies in the appraisal of diabetes-related foot disorders - Time to Upgrade
• Physicians may underestimate the impact of minor hypoglycaemic events associated with insulin treatment in type 2 diabetes. Results from the Global Attitude of Patients and Physicians (GAPP) Online Survey

CPD ACCREDITED DIABETES TRAINING
A theme common to several of the pieces in this issue revolves around prevention. Whether it is the prevention of hypoglycaemia, the early identification and thus prevention of severe diabetic ketoacidosis or the prevention of bone fractures, our accomplished authors write of something that is essentially fundamental to the practice of diabetes medicine. Prevention can often come with an upfront price tag given the yet intangible costs of a future event. Sean Pincus, who writes on the role of novel technologies in foot care, makes the point that the cost of inertia is in itself a problem. Much akin to the plethora of new drugs available for the glycaemic management of a person with diabetes, it is refreshing to note the many additional technologies available to the podiatrist. Sean makes the point that we should not diminish the value of solid history taking and the performance of a detailed clinical examination, and thereafter and where appropriate, then harness these new applications.

Adri Kok reminds us that we often underutilize bone densitometry in diabetes care. People with diabetes are, for various reasons, predisposed to the development of osteoporosis and bone fractures. We should remain mindful that both men and women need to be screened for this condition where appropriate. In a similar vein, paediatric endocrinologist David Segal highlights the early identification of diabetic ketoacidosis and the often-lethal consequences of either delayed presentation or inappropriate treatment. It is also refreshing to be able to include a topic applicable to type 1 diabetes. The pendulum then swings in the opposite direction in terms of hypoglycaemia and the associated negative consequences that are now being identified as part of this problem.

Finally, the launch of this Issue coincides with the 16th Annual CDE Postgraduate Forum in Diabetes Management. I hope that each attendee gains much value from participating in addition to using their time to network with colleagues. I am grateful to Sub-Editor Michael Brown who has worked exceptionally hard on ensuring this Issue ‘gets to the press on time’, whilst also playing a fundamental role in putting the Forum together.
“Treatment without prevention is simply unsustainable.”
Bill Gates

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Osteoporosis and bone health in Diabetes

Introduction
Bone health has never been a specific focus area in the management of either type 1 or type 2 diabetes. However, it has become clear that if patients with type 2 diabetes develop complications due to inadequate glucose control, they may have a higher risk of suffering osteoporotic fractures. Similarly, people with type 1 diabetes typically have lower bone mineral density (BMD) and are therefore at an increased risk of fractures. Diabetes-related osteoporosis may be associated with additional co-morbidities such as smoking and obesity.

With an increasing life expectancy documented for many people with diabetes, previously undetected osteoporosis may be fast emerging as another condition worthy of intensive investigation and intervention.

Epidemiology
In South Africa, the prevalence of osteoporosis in the Caucasian, Asian and mixed-race groups is similar to that of developed nations. No specific fracture data exist for South Africa, and although osteoporosis of the hip is less prevalent in the African population, vertebral bone mass appears to be similar in black and white South Africans. Osteoporosis is not a benign condition, and is associated with a significant morbidity and mortality. With an ageing population and increased life expectancy (at present only 7.5 million people are over the age of 50) and with an increase in diabetes incidence and prevalence, the incidence of osteoporosis and osteoporotic fractures will increase. A grim picture for future osteoporosis and its associated fracture risk (See Table 1) is thus fast emerging.

Pathophysiological mechanisms
Various mechanisms for osteoporosis in diabetes have been proposed and investigated. An association exists between decreased BMD and microvascular complications. Data from diverse populations such as postmenopausal women and children with longstanding type 1 diabetes have confirmed this. Typically, diabetes complications develop because of poor glucose control, a genetic predisposition and existent co-morbidities. This cannot be linked to exogenous insulin...
administration, since insulin does not cause bone loss, but an autoimmune or inflammatory mechanism early in the disease process of diabetes may be implicated. Furthermore, diabetes impairs normal osteoblast functioning, although, curiously, not all sub-optimally controlled patients have established osteoporosis. Lower levels of insulin-like growth factor (IGF-1) found in the context of type 1 diabetes may play a role. This anabolic hormone usually maintains healthy bone formation.

Hyperglycaemia, with a resultant increase in oxidative stress, adversely affects osteocalcin production and the downstream signalling pathways with a resultant imbalance between osteoblast and osteoclast activity. Bone fragility will increase the risk of fractures.

Other factors adversely influencing affecting bone quality include hyperinsulinaemia, deposition of advanced glycation end products (AGEs) in collagen, hypercalciuria, renal failure, microangiopathy and inflammation.

Certain medications could also be implicated in the pathogenesis of osteoporosis. Loop diuretics used in the treatment of hypertension and cardiac failure may result in increased urinary loss of calcium. In contrast, thiazide diuretics decrease the loss of calcium.

**Table 1: Risk factors for osteoporotic fractures in Diabetes**

<table>
<thead>
<tr>
<th>Risks for osteoporosis</th>
<th>1) Diabetes</th>
</tr>
</thead>
<tbody>
<tr>
<td>a) Type 1 diabetes</td>
<td></td>
</tr>
<tr>
<td>b) Sub-optimal glycaemic control</td>
<td></td>
</tr>
<tr>
<td>c) Nephropathy</td>
<td></td>
</tr>
<tr>
<td>2) Complications of diabetes</td>
<td></td>
</tr>
<tr>
<td>a) Neuropathy</td>
<td></td>
</tr>
<tr>
<td>b) Diabetes related diarrhea</td>
<td></td>
</tr>
<tr>
<td>3) Conditions often associated with diabetes</td>
<td></td>
</tr>
<tr>
<td>a) Grave’s disease</td>
<td></td>
</tr>
<tr>
<td>b) Coeliac disease</td>
<td></td>
</tr>
<tr>
<td>c) Amenorrhoea</td>
<td></td>
</tr>
<tr>
<td>d) Delayed puberty</td>
<td></td>
</tr>
<tr>
<td>e) Eating disorders</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Risk for falls</th>
<th>1) Recurrent hypoglycaemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>2) Nocturia</td>
<td></td>
</tr>
<tr>
<td>3) Reduced visual acuity, retinopathy, cataracts</td>
<td></td>
</tr>
<tr>
<td>4) Poor balance due to neuropathy, foot ulcers and amputations</td>
<td></td>
</tr>
<tr>
<td>5) Orthostatic hypotension</td>
<td></td>
</tr>
<tr>
<td>6) Impaired joint mobility due to arthritis and the ‘diabetic foot’</td>
<td></td>
</tr>
</tbody>
</table>


Other factors adversely influencing affecting bone quality include hyperinsulinaemia, deposition of advanced glycation end products (AGEs) in collagen, hypercalciuria, renal failure, microangiopathy and inflammation.

Thiazolidinediones (TZD’s) may interfere with bone regulation, specifically osteoblasts and adipocytes, via action on PPAR-γ. TZD’s switch mesenchymal progenitor cell development to adipose rather than bone tissue.

Metformin has a direct protective effect on bone tissue by reducing AGE accumulation, whereas insulin acts directly on improving osteoclast activity.

Incretin mimetics are new treatment options for people with type 2 diabetes. Emerging data from their use reveals that they may in fact reduce fractures.

The use of anti-retroviral therapy has improved longevity, but the sometimes-negative side effect of these medications on bone health requires further study.

**Management strategies**

1. **Nutrition**

A diet rich in calcium and Vitamin D is recommended. Low-fat dairy products and dark green leafy vegetables are good sources. Vitamin D is a steroid hormone with a range of functions in skeletal and non-skeletal tissues. Specifically, it plays an important role in calcium absorption and bone health. It promotes bone formation by increasing calcium and phosphate in plasma, regulating osteoclast and osteoblast activity and it inhibits hyper secretion of parathyroid hormone (PTH).

Vitamin D may inhibit fat accumulation, increase insulin synthesis and preserve pancreatic islet cells, decrease insulin resistance and reduce hunger. Low serum vitamin D levels are not in themselves a cause of obesity, but many obese people have vitamin D deficiency. At present, evidence does not support supplementation of vitamin D to prevent or treat obesity nor type 2 diabetes.

2. **Physical Activity**

Bone is living tissue and responds to exercise by becoming stronger especially in response to weight bearing. Exercise furthermore, prevents bone loss, enhances
balance and flexibility and decreases the likelihood of falling and of fractures. Exercise improves insulin action by activating insulin receptors in muscle.

3. Healthy Lifestyle

Smoking is bad for bones. It may result in earlier menopause in females with earlier bone loss. Smoking also reduces dietary calcium absorption. Alcohol consumption promotes poor nutrition, bone loss and increases the risk of falling.

Low bone mass may result from other factors such as female sex, Caucasian ethnicity, low body mass index (BMI < 19 kg/m²) and a maternal or personal history of fractures. Patients with type 2 diabetes have an increased BMD, but this does not predict bone quality - dual-energy X-ray absorptiometry (DEXA) screening does not assess qualitative bone changes. There is a higher risk of falls in those with microvascular complications. Typically, this exists in those with established peripheral neuropathy. Hypoglycaemia, especially in those with reduced warning signs, also raises the risk of falls, as does nocturia, visual impairment and pre-existing neurological conditions.

Treatment

Required essentials in the management of osteoporosis in the person with diabetes are optimal glycaemic control, adequate intake of vitamin D, DEXA scanning to screen for low BMD in those at risk and the prevention and treatment of complications of diabetes.

The pharmaceutical treatment of established osteoporosis is summarised in Table 2.

Conclusion

Osteoporosis is a heterogeneous syndrome with few head-to-head studies comparing the efficacy and safety of medications. Treatment should be highly individualized and must include assessment of the risk of future fracture and falls.

The person with either type 1 or type 2 diabetes typically has unique additional risks for the development of osteoporosis. We need to acknowledge this and integrate the identification and treatment of this condition into our regular care protocols.

REFERENCES AVAILABLE ON REQUEST

### Table 2: Therapeutic options in treating osteoporosis

<table>
<thead>
<tr>
<th>Class</th>
<th>Drug</th>
<th>Typical dose</th>
<th>Potential adverse effects</th>
<th>Increased spine BMD (Duration of treatment)</th>
<th>Vertebral fracture reduction</th>
<th>Hip fracture reduction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hormone therapy</td>
<td>Oestrogen + progesterone</td>
<td>0.625/2.5 mg PO</td>
<td>• Venous thromboembolism • Breast cancer</td>
<td>5-7 % (2 years)</td>
<td>+++</td>
<td>++</td>
</tr>
<tr>
<td>Bisphosphonates</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. Alendronate</td>
<td></td>
<td>70 mg/week PO</td>
<td>• Oesophagitis • Renal failure • Creatinine clearance &lt; 30 • Abdominal pain • Dyspepsia • Atypical fractures • Contra-indicated renal failure</td>
<td>5-6 % (2-3 years) 4-5 % (1.5-3 years) N/A</td>
<td>+++</td>
<td>++</td>
</tr>
<tr>
<td>2. Risedronate</td>
<td></td>
<td>35 mg/week PO</td>
<td></td>
<td></td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>3. Zoledronate</td>
<td></td>
<td>5 mg/100 ml Infusion Annually</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Increased Bone formation + Decreased Bone resorption</td>
<td>Strontium ranelate</td>
<td>Sachet 2g daily PO</td>
<td>• Diarrhoea • Nausea • Use with care in CCF • Severe renal impairment • Hypersensitivity reaction</td>
<td>Significant continuous ↑ BMD (5-10 years)</td>
<td>+++</td>
<td>++++</td>
</tr>
<tr>
<td>Anabolic Hormone</td>
<td>Parathyroid Teriparatide</td>
<td>20 μg subcutaneously</td>
<td>• Arthralgias • transient orthostatic hypotension • CR in nephrothiazis</td>
<td>9 % (18 months)</td>
<td>+++</td>
<td>Non-vertebral combined ++</td>
</tr>
<tr>
<td>Calcium regulation hormone</td>
<td>Calcitonin</td>
<td>200 IU nasal spray</td>
<td>• Rhinitis</td>
<td>1-3 % (5 years)</td>
<td>+</td>
<td>No</td>
</tr>
<tr>
<td>Selective serotonin receptor modulator</td>
<td>Raloxifene</td>
<td>6 mg day</td>
<td>• Venous thromboembolism • Hot flushes</td>
<td>2-3 % (2-3 years)</td>
<td>+++</td>
<td>No</td>
</tr>
</tbody>
</table>

The Chain of DKA Management - what can go wrong and how do you fix it?

A fter the dangers and the turmoil of an initial diagnosis of type 1 diabetes, follows a long road with many possible bumps and bruises. Good education, preparedness and early and appropriate interventions can reduce the occurrence of severe diabetic ketoacidosis (DKA). Evidence based management has been shown to reduce the morbidity and mortality associated with severe DKA and ward off life threatening cerebral oedema, a complication of 1 percent of DKA admissions. This review will focus on the latest evidence based recommendations and how to incorporate them into your practice.

The most effective mechanism to reduce mortality associated with DKA is to prevent DKA. DKA most commonly presents in children newly diagnosed with type 1 diabetes (T1DM). Systematic reviews of DKA at presentation prove that time to diagnosis is a major risk factor. On average, children have osmotic symptoms of diabetes (polyuria, polydipsia, weight loss and new-onset inappropriate enuresis) for 2-3 weeks before diagnosis and have seen a health care professional at least once in the week before diagnosis.

The frequently encountered symptoms of DKA such as nausea and vomiting, and rapid respiration can result in diabetes commonly misdiagnosed as gastroenteritis or pneumonia. These pitfalls can be avoided if a detailed history is taken, including enquiry into any symptoms of diabetes. DKA typically presents with abdominal pain, nausea, and vomiting without diarrhoea, while the combination of diarrhoea and vomiting is more likely to be present in gastroenteritis. The rapid breathing and shortness of breath associated with pneumonia is typically associated with fever, cough, wheeze or upper respiratory tract signs. Abdominal pain may be misdiagnosed as appendicitis. In adults, DKA is often precipitated by urinary tract infections, pneumonia, myocardial infarctions and strokes.

Publicity campaigns highlighting the signs and symptoms of diabetes have had mixed but overall positive results. The most effective to date was a publicity campaign targeting primary and secondary schools and paediatricians in Parma, Italy. They managed to reduce the cumulative frequency of DKA admissions in new-onset T1DM from 78 % to 12.5 %. In the 2 years following the campaign, no DKA admissions were recorded and a positive impact was maintained for 10 years.

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While initial presentation of diabetes accounts for the greatest number of DKA admissions, re-admission in patients with known diabetes accounts for the balance. It is in this area that sick day education, preparedness, access to a 24-hour ‘hotline’ and early appropriate intervention can prevent admission to hospital.

DKA is the result of an absolute or relative deficiency of insulin. An absolute deficiency is typically present in newly diagnosed patients. Insulin deficiency results in the activation of a cascade of metabolic events that, if not interrupted, will result in worsening acidosis, dehydration and possibly even death. Insulin’s primary function is to store incoming carbohydrates and drive peripheral glucose utilization. Insulin also suppresses glucagon release and endogenous hepatic glucose production and prevents lipolysis. In its absence, hepatic glucose production runs amok and osmotic diuresis follows, resulting in the progressive development of dehydration. Unchecked lipolysis dumps ever-increasing amounts of free fatty acids into the system as ketone precursors. The primary ketones produced are beta hydroxybutyrate (BOHB), acetoacetate (AcAc) and acetone. As the blood pH drops, and dehydration progresses, a surge in counter regulatory hormones (cortisol, growth hormone and epinephrine) accelerate the delivery of gluconeogenic precursors to the liver, adding even more fuel to the fire.

As the ketone levels climb, they begin to cause symptoms of abdominal pain and nausea. Ketones activate the chemoreceptor trigger zone in the brain with resulting emesis. Acetone, a volatile ketone, can be smelled on the breath as a fruity odour. The other two ketones, BOHB and AcAc, usually present in a resting state in equimolar concentrations, start to climb differentially. BOHB, the primary blood ketone, rises to levels 3-5 times greater than AcAc. Early detection of climbing blood ketones has become the most sensitive measure of developing ketoacidosis and has been recommended to be included in the primary definition of DKA. The increasing availability of point of care blood ketone meters (Abbott Freestyle Optium) is a huge advance in early detection and management of ketonaemia.

Acetoacetate measured on urine dipsticks has many obvious limitations that make blood ketone testing superior. AcAc testing is dependent on a chemical reaction on a urine test strip. Many episodes of DKA admissions occur because ketones were not checked in a timely manner or at all. The test requires a person with diabetes to collect a urine sample and perform the simple test. The activation energy required to collect the sample in the first place is often not mustered, especially in people feeling ill, perhaps dehydrated and struggling to pass urine. It is especially difficult in young children who have not been potty trained. It is also a time-dependent test requiring the production of urine in the first place. Blood ketone testing is quick, has very little activation energy, gives a result that is easy to interpret and is open to repeat testing at will, providing information on the relative improvement or worsening of the clinical situation.

A randomized study compared blood ketone testing versus urine ketone testing in patients with known diabetes, over a period of 578 sick days and 21,548 days of follow-up. Those given blood ketone meters were significantly more likely to check for ketones than were those in the urine testing group (90.8% vs. 61.3%). The subsequent incidence of hospitalization was also dramatically reduced in the blood ketone-testing group, with 38 admissions per 100 patient years, versus 75 in the urine ketone-testing group.

Patient education on what to do with high blood glucose values and sick day management can lead to early detection of ketones and timely intervention to control the situation and prevent progression to DKA or hospitalization. To do this, patients need to know to test for ketones if:
• their blood glucose is unusually elevated above their usual range or above 15 mmol/l for consecutive tests
and
• if any symptoms of ketones are present such as abdominal pain, nausea or vomiting, or any symptoms of illness.

Here the application of a standard “Coke and insulin” protocol can switch off ketone production and halt progression to DKA. The oral fluids are used to replace fluids lost through increased urine production. Sipping on roughly 250 ml per hour over an
hour reduces the chances of vomiting and adequately replaces fluids. Injecting fast acting analogue insulin at 0.1 IU/kg/hour will switch off ketone production and clear the existing ketones. Initially, sugar free fluids should be consumed if the blood glucose is high enough to tolerate the increased insulin delivery. If the blood glucose is below 15 mmol/l or drops below 15 mmol/l during ketone treatment, sugar-containing fluids should be consumed to allow ongoing insulin injections while preventing blood glucose levels from dropping too low. Typically, 1 to 3 hours of injections will halt ketone production and avoid progression to DKA. For this to work patients need education on sick day management and have the ability and the supplies needed to do home ketone testing.

Progression to DKA
A study comparing serum bicarbonate and BOHB levels in 129 hospitalized children with DKA, found that a blood ketone level of 3.0 mmol/l corresponded to a serum bicarbonate level of 18, while a blood ketone level of 4.4 mmol/l correlated with a serum bicarbonate of 15 mmol/l. This evidence suggests that given the other confounding factors in pH and serum bicarbonate analysis at presentation, a serum ketone measurement may be very useful for evaluating patients with suspected DKA. In fact a blood ketone level >3 mmol/l has been added to the diagnostic criteria for DKA, along with a venous pH < 7.3, a serum bicarbonate < 15 mmol/l and a blood glucose level >11 mmol/l.

Laboratory BOHB measurements correlate very well with point of care BOHB measurement below a blood ketone level of 4 mmol/l. Because any meter value >1.5 mmol/l indicates significant ketosis and potential for DKA, one can recommend the use of point of care devices not only for the initial diagnosis but also during ongoing monitoring and management of DKA. BOHB values decline rapidly after initiation of treatment, far faster than urine AcAc measurements, which can remain elevated for hours into therapy. Currently studies suggest that patients can be transitioned to subcutaneous insulin and oral intake once the pH is >7.3 and 2 consecutive BOHB measurements are <1.0 mmol/l.

DKA is a metabolic emergency requiring prompt diagnosis, appropriate protocol driven management and regular monitoring to document improvement and detect complications early. One of the most dreaded complications of DKA is cerebral oedema, which complicates 1 % of DKA episodes. It has a high mortality and survivors often have persistent neurological deficits. Other causes of death include sepsis, shock and electrolyte abnormalities.

Treatment of DKA involves fluid resuscitation and replacement, electrolyte replacement and insulin therapy. The goal is to relieve acidosis, extinguish ketone body formation and slow normalization of glucose levels, all while preventing complications of treatment or worsening of the underlying condition. A glucose-centric approach often leads to complications.

Too much fluid and too much insulin, and a sicker patient, spells trouble.

Urine ketone testing is also falling out of favour because of limitations due to false positive and false negative results. Urine ketone tests using nitroprusside reagents give false positive results in the presence of a number of drugs containing sulphhydryl groups (see table below). False negative results are more dangerous. Prolonged exposure of the strips to air or expired strips will give a false negative result, resulting in underestimation of the severity of the clinical situation and delayed intervention. Very acidic urine specimens such as after the consumption of high doses of ascorbic acid, a commonly ingested over the counter medication, also causes false negative results.

Table 1: Causes of false results in the urine testing for ketones

<table>
<thead>
<tr>
<th>False Positive</th>
<th>False Negative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cefixime (antibiotic)</td>
<td>Vitamin C (high dose)</td>
</tr>
<tr>
<td>Tricyclic antidepressants</td>
<td>Old urine</td>
</tr>
<tr>
<td>Captopril (ACE inhibitor)</td>
<td></td>
</tr>
<tr>
<td>Valproic acid (anti-epileptic drug)</td>
<td></td>
</tr>
<tr>
<td>High dose glucocorticoids</td>
<td></td>
</tr>
<tr>
<td>Levodopa (high dose)</td>
<td></td>
</tr>
<tr>
<td>Mesna (chemotherapy adjunct)</td>
<td></td>
</tr>
<tr>
<td>N-Acetylcysteine (mucolytic)</td>
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</table>

On admission, a thorough history and physical examination should be performed. Look for precipitating factors such as sepsis, the use of recreational or prescription drugs (especially glucocorticoids), stroke, myocardial infarction, urinary tract infection or insulin omission. Carefully examine for and document neurological status, including pupil size and the Glasgow Coma Score (GCS), and the presence of headache and lethargy. Importantly, continue to monitor and document the situation regularly and repeatedly until resolution.
Continuous heart rate monitoring is essential to detect early signs of cerebral oedema, such as relative bradycardia. Make sure that emergency drugs are present and protocols written up for insulin, glucose management, and detection and management of cerebral oedema. Use a bedside neurological evaluation protocol to detect cerebral oedema.

**Fluid management**

Clinical estimates of hydration status are notoriously inaccurate. Given that over-hydration (too much fluid and given too quickly) can cause major problems, it is better to be conservative. The average patient is 8% dehydrated. For calculation of maintenance and rehydration, one could work on 5% dehydration for those with mild acidosis (venous pH 7.1-7.3). Use 10% for those with a pH < 7.1. Aim to rehydrate slowly over 48 hours. An initial fluid bolus of 20 ml/kg normal saline or Ringers Lactate is appropriate. The initial fluid resuscitation is important and should be completed before any insulin is administered. Maintenance fluids should contain a minimum of 0.45% sodium chloride and up to 0.75% sodium chloride and be given at a rate of 1.5 to 2 times maintenance. Add glucose to obtain 5-10% glucose/dextrose solutions to keep blood glucose stable while infusing adequate amounts of insulin.

**Electrolytes**

**Potassium**

Potassium is the most dangerous electrolyte - often low-to-normal on admission, it is expected to drop dangerously during therapy. For every 0.1 increase in pH, the K⁺ level drops by 0.6 mmol/l. Insulin also drives potassium from the intravascular space back into the cells. Potassium should be replaced early in the first bags of maintenance fluids. Concentrations of 20-40 mEq/l, and even higher, may be required.

**Sodium**

The high glucose levels lead to an increase in serum osmolarity and a water shift from the intracellular to the intravascular space causing dilutional ‘hyponatraemia’. It is prudent to calculate the corrected serum sodium:

\[\text{Corrected Na}⁺ (\text{mmol/l}) = \text{measured serum Na}⁺ (\text{mmol/l}) + \frac{\text{blood glucose (mmol/l) – 5}}{5} \times 1.6\]

As the glucose levels drop during therapy, the measured Na⁺ should increase but the Corr Na⁺ should remain the same. A fall in Corr Na⁺ may indicate over hydration with hypotonic solutions and increases the risk of developing cerebral oedema.

**Bicarbonate (HCO₃⁻)**

Serum bicarbonate levels correlate reasonably well with the pH. Bicarbonate administration has been associated with an increased risk of developing cerebral oedema and the need for K⁺ replacement. Given the increased risks and lack of evidence to support its use in the management of DKA, it is probably best avoided.

**Insulin**

Insulin is the key to switching off ketogenesis and resolving the acidosis. However, care must be given because too much insulin can cause harm. Continuous insulin infusions are favoured and bolus insulin is not recommended. It is best to start insulin after fluid resuscitation has commenced and probably best if given after the first hour of fluids. Evidence is emerging documenting equivalent DKA resolution on low dose insulin infusions. For moderate DKA (pH 7.1 to 7.3) 0.05 IU/kg/hour is adequate but 0.1 IU/kg/hour is probably still superior in severe DKA (pH < 7.1).

Insulin binds to plastic and filters in the infusion tubing. Preferably, mix 200 IU of rapid acting insulin in a glass 200 cc normal saline bottle for infusions (1 IU/cc). Filters should not be used.

Excessive insulin use has been associated with a rapid fall in serum osmotic pressure due to rapid decline in serum glucose. This can drop cerebral perfusion pressures and increase the risk of developing cerebral oedema.

Subcutaneous injections of rapid-acting insulin analogues have been used to treat mild to moderate DKA when continuous infusions are not an option.

Some protocols reduce insulin doses according to a ‘sliding scale’ as blood glucose levels decline. The secret of DKA management is to provide enough insulin to clear ketosis. It is best to start supplementing glucose in the maintenance fluids rather than reducing insulin to keep blood glucose levels within a target range of 4 to 12 mmol/l.

**Investigations and monitoring**

On admission, a series of blood tests is required. A venous blood gas (VBG) is adequate to obtain pH information and limits the risks associated with arterial access. A venous pH is only 0.03 different from an arterial pH. The VBG often also gives a rapid estimate of electrolytes, HCO₃⁻ base excess and pCO₂. A serum BOHB can be measured at the bedside for baseline and monitoring. A U+E (urea and electrolytes test) and glucose gives information that is more accurate on electrolyte status. It can be used to calculate the serum osmolarity and corrected sodium. A full blood count often shows an increase in white cell count with a left shift that is not associated with infection. C-reactive protein (CRP) can be elevated too. Cardiac enzymes, blood and urine cultures and other investigations may be required as clinically indicated.

**Complications**

The most dreaded complication of DKA is cerebral oedema occurring in 7 per 1000 episodes of DKA. Subclinical cerebral oedema is much more common occurring in up to 19% of
Health benefits of weight reduction in patients with diabetes

Overweight and obesity account for about 80-90% of all cases of type 2 diabetes and are important obstacles to the successful long-term management of diabetes.

The underlying metabolic abnormalities of diabetes are the result of obesity and predispose patients to hypertension and cardiovascular disease. Weight reduction is an important goal in treating type 2 diabetes and some consider it the cornerstone of diabetes therapy. Not only does it result in physiological benefits, it also increases longevity.

Modest weight loss (5-10%) provides the most striking benefits:
- Increases life expectancy of overweight patients with type 2 diabetes by 3-4 years
- Reduces diabetes-related deaths by more than 30%
- Fall of up to 50% in fasting glucose in those newly diagnosed with diabetes
- 40-60% fall in incidence of diabetes for people at risk, such as those with impaired glucose tolerance

Achievement and maintenance of ideal body weight may reduce the need for glucose-lowering medications, including insulin, and lower the cost of diabetes medication. Effective interventions for weight management should commence as soon as diabetes is diagnosed (or at the diagnosis of impaired glucose tolerance or abdominal obesity). It has been demonstrated clinically that people with type 2 diabetes experienced more difficulties losing weight than overweight people without diabetes. Failure to achieve and maintain weight loss may not be due to non-adherence, but rather due to the altered metabolism of diabetes. Added to this is the fact that too many ‘diets’ focus on improvement of blood glucose and lipids, rather than a focus on weight management.

In a survey done of specialist physicians who treat obesity, phentermine ranked as the most prescribed anti-obesity medication. Duromine (phentermine) is a sustained action anorectic agent used in obese patients as a short-term adjunct in a medically monitored comprehensive regimen of weight reduction based, for example, on exercise, dietary interventions and behaviour modification.

With Duromine, patients can conservatively expect to achieve an average weight loss of 1.19 kg per week in the first 4 weeks and 0.56 kg per week in weeks 5-12. Faster initial weight loss is associated with a greater likelihood of achieving 10% weight loss and preventing weight regain in the long-run.

Duromine in conjunction with dietary interventions and exercise helped patients with obesity and type 2 diabetes achieve early and significant weight loss.

Gershberg et al demonstrated that patients lost significantly more weight with Duromine than on diet alone and also achieved significant reductions in blood pressure and serum cholesterol.

Duromine™

Phentermine
50 years real world clinical experience

Dosage and directions for use
- 15 mg or 30 mg capsule once daily at approximately 7 a.m.

**Duromine is not indicated in children under 16 years old.

References:

Scheduling status: Proprietary name (and dosage form): Duromine™ 15 mg and 30 mg capsules. Compositions: Sustained action ion-exchange resinate granules, available as capsules containing phentermine 15 mg and 30 mg. Pharmaceutical classification: A 1.3 Anorexigenics. Reference number: 15 mg B6673; 30 mg B6954 [Art 10517963]. Name and business address of applicant: Nova Pharmaceuticals Ltd, C/o Reg. No. 196/2004/G07, The Ralway Road, Bedfordview. Tel: No. 511, 087 0020. www.novapharma.co.za. For full prescribing information, refer to the package insert approved by the medicines regulatory authority. Further information is available on request from Nova Pharmaceuticals, IN35173.
DKA admissions. Due to rarity of cerebral oedema, studies on aetiology and pathogenesis are limited. The current purported mechanisms of cerebral oedema centre around disruption of the blood-brain barrier and the development of vasogenic oedema. A component of cytotoxic cerebral oedema may well be a contributing or late factor. Inflammation and reperfusion injuries probably both contribute. The peak incidence of cerebral oedema occurs between 4 and 12 hours into therapy but can occur up to 24 hours after admission.

Studies suggest that certain patient characteristics and therapy factors may increase the risk. These are younger age, newly diagnosed patients, severity of the acidosis at presentation, higher urea levels, lower PaCO₂ levels, administration of bicarbonate, larger volumes of fluid over the first 3-4 hours, lower plasma sodium but no difference in fluid sodium concentration, administration of insulin within the first hour of fluid treatment (OR 4.7) and bolus insulin administration.

Anticipation of cerebral oedema by identifying risk factors and using a neurological testing protocol may allow for early detection and management to reduce the occurrence and severity of cerebral oedema events. An evidence-based protocol for bedside neurological evaluation has been proposed. The presence of one diagnostic criterion, two major or one major and two minor criteria had a sensitivity of 92% in predicting cerebral oedema.

At the earliest sign or symptom, therapy with either mannitol or hypertonic saline should be instituted. Currently 3 ml/kg of 5% sodium chloride administered over 5-10 minutes is the favoured first line therapy. Should symptoms not rapidly resolve, 5 ml/kg (1 g/kg) of 20% mannitol should be infused over 15 minutes. If mannitol is used first without response, hypertonic saline should be used next. CT scanning should be performed after intervening to look for other causes of neurological deterioration that may benefit from specific treatment.

**Transition to subcutaneous insulin**

Once DKA has resolved to the point that the pH is >7.3, the HCO₃ >15 and BOHB on 2 consecutive occasions is < 1 mmol/l the patient can be transitioned to subcutaneous insulin and meals re-instated. Low levels of ketones may persist for another one to two days but should not prolong hospitalization. The author does not recommend attempting to obtain blood glucose stability in the hospital setting. This tends to prolong hospitalization unnecessarily, increase costs and inflicts greater psychological trauma on the patient. The logistical, dietary and physiological changes inherent in the hospital setting make this a near impossible task and the patient is better off if discharged into the care of the family and an experienced diabetes care team. Outpatient education, and initiation and intensification of insulin are ideal. Counselling and re-education of existing patients presenting in DKA can help to reduce future admissions.

**Take home points**

1. Educate communities about the symptoms of diabetes to facilitate early diagnosis and DKA prevention.
2. Educate patients on sick day protocols, including insulin administration and ketone testing.
3. Use blood ketone measuring devices in young children, patients using insulin pump therapy and, if possible, all ketosis-prone patients with diabetes.
4. Use evidence-based, protocol driven therapies for DKA management.

**REFERENCES AVAILABLE ON REQUEST**

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**Table 2: Bedside evaluation of neurological state of children with DKA**

<table>
<thead>
<tr>
<th>Diagnostic criteria</th>
<th>Major criteria</th>
<th>Minor criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Abnormal motor or verbal response to pain</td>
<td>Altered mentation/fluctuating level of consciousness</td>
<td>Vomiting</td>
</tr>
<tr>
<td>Decorticate or decerebrate posture</td>
<td>Sustained heart rate deceleration (decline more than 20 bpm or &lt; 70% of maximum) not attributable to improved intravascular volume or sleep state</td>
<td>Headache</td>
</tr>
<tr>
<td>Cranial nerve palsy (especially III, IV, and VI)</td>
<td>Age-inappropriate incontinence</td>
<td>Lethargy or being not easily aroused from sleep</td>
</tr>
<tr>
<td>Abnormal neurogenic respiratory pattern (e.g., grunting, tachypnoea, Cheyne-Stokes respiration, apneusis)</td>
<td></td>
<td>Diastolic blood pressure &gt;90 mmHg</td>
</tr>
</tbody>
</table>

**Signs that occur before treatment should not be considered in the diagnosis of cerebral oedema**

Adapted from Diabetes Care, Volume 27, Number 7, July 2004
With the introduction of any new routine like a new job, a new environment, or a new relationship into a person’s life, that person has to adapt and assimilate the change into his or her everyday living. If perception of the change is negative, assimilation is much more difficult to process and achieve. Many people perceive the introduction of insulin injections as a negative change.

It should not be surprising then that 27% of children (Simmons et al, 2007) and 33% of adults with type 2 diabetes (AADE, 2008) introduced to insulin injections, were resistant to taking insulin. This group of people approach their injections with a sense of dread and say that injections are the hardest part of managing their condition. The fear can be severe enough to prevent them from administering insulin at all. One survey found that more than half of those ‘resistant’ people skipped injections from time to time.

Psychological Insulin Resistance (PIR) is one name given to psychological barriers to the initiation of insulin therapy. Most of the various studies done on this subject, define PIR as reluctance to initiate insulin treatment. However, about a quarter of them also consider PIR to include insulin injection omission (Gherman et al, 2011). Of interest is that barriers to insulin initiation are found in health care providers as well (Peyrot et al, 2005). PIR has long-term consequences, since without injections, diabetes cannot be effectively controlled and the ‘resistant patient’ is at risk of developing complications sooner.

Reasons why people resist insulin injections

1. Anxiety

A child or teenager newly diagnosed with diabetes will experience a range of emotions including shock, denial, anger, sadness, fear and guilt.

Fear or anxiety is usually related to

- fear of needles and multiple injections (including needle phobia)
- fear of being overwhelmed by injecting and other tasks that have to be performed daily
- fear of hypoglycaemia after their first experience of such
- feeling like they are a burden on the family, especially if there are financial concerns
A cross-sectional survey of 35-60 year old Dutch patients who were treated with insulin (58 % with type 1 diabetes) was done to ascertain whether they had PIR or not, and if so, to understand why. Patients with extremely high scores on the Diabetes Fear of Injection and Self-testing Questionnaire (D-FISQ) had higher levels of depression, and fear of hypoglycaemia. 11 % of these patients had major depression (Mollema et al, 2001).

Elderly people find it very difficult to assimilate any new treatment, but more particularly insulin. Many of them have extreme PIR. Bahrmann et al (2014) examined PIR in elderly patients aged 70-90 years and found they have a significantly more negative attitude towards insulin. Their main reasons were fear of injections and fear of stigmatisation. They also expected disadvantages from insulin treatment. Many rated the clinical efficacy of insulin as low.

A large clinical study of type 2 patients found that 27 % of patients initially refused treatment with insulin and in another study of 708 patients with type 2 diabetes, 28 % reported that they would not take insulin even if it were prescribed by their physician (Peyrot et al, 2005; Polonsky et al 2008). 50 % of these were unwilling because they are afraid of needles or injection pain.

Other reasons given for insulin refusal were fear of weight gain, fear of dependence, depression, lack of knowledge, and fear that it would interfere with their lifestyle (Gherman et al, 2011). Many are afraid of hypoglycaemia especially if they have experienced it previously.

2. Needle phobia

The DSM-IV lists needle phobia as a “marked and persistent fear that is excessive or unreasonable, cued by the presence or anticipation of a specific object or situation (e.g. receiving an injection)”. About 10 % of people in the general population have needle phobia (Diabetes UK 2014). Needle phobia is a great deal more than just ‘not liking injections’. It is important that it is recognised rather than making assumptions that the person is just being ‘non-compliant’ or ‘not willing to do what they should’. Needle phobia needs treatment - medical aids have an International Statistical Classification of Diseases and Related Health Problems (ICD-10) code for the condition. According to James Hamilton (APA, 2012) “the aetiology of needle phobia lies in an inherited vasovagal reflex of shock, triggered by needle puncture. Individuals who inherit this reflex learn to fear needles though successive exposures to needles.”

A study in Denmark (1997) found that 8.3 % of young people between 6 and 19 years old with diabetes classed themselves as having pronounced needle phobia. They found that all family members in that group had a negative attitude towards diabetes and that 16-17 % of the parents of these children had needle phobia themselves. They found a clear relationship between needle phobia, injection pain, self-testing pain and attitude towards diabetes.

Mollema et al (2001) noted that 11 % of those tested had extreme PIR. Extreme PIR probably reflects needle phobia. This group reported higher levels of anxiety, major depression, diabetes-related distress, lower levels of general well-being, and poor glycaemic control.

9 % of 33 adult patients surveyed at the CDE Central Office in Houghton, using questions from the D-FISQ on how they felt about having injections, appeared to have extreme PIR or needle phobia. 7 % of 30 youth patients with type 1 diabetes had needle phobia. 23 % said they sometimes avoided injections and/or testing. 37 % said they hated finger-prick testing but did it anyway.

Needle phobia is likely to compromise glycaemic control as well as emotional wellbeing. Likewise fear of finger pricks can be a source of distress and will hamper self-management.

3. Patient Beliefs

Some patients believe that needing insulin treatment is a sign that their diabetes has become much worse. They often blame themselves because they believe they should have taken better care of their bodies and their diabetes. They experience a sense of failure. Many will promise to improve their ‘diet’ and do more exercise to avoid going on insulin. They see insulin treatment as a ‘last resort’.

Some people associate insulin therapy with complications in close relatives. For example, a 69-year-old woman who has type 2 diabetes and is eligible for insulin treatment, has a brother who has type 1 diabetes with many complications. She believes that the insulin caused the complications and begged to be given time to try harder to get her blood glucose levels down (with exercise and better eating) before starting insulin.

Polonsky and Jackson (2004) found that 61 % of patients at the Joslin Centre for Diabetes resisted insulin treatment because they believed that once they start insulin, they could never stop and that would mean they had little control over their lives. 50 % believed that insulin therapy would restrict their lives so that they could no longer have adventures or be spontaneous. They also found that 40-50 % of patients believed that they could not handle the demands of insulin therapy.

Some insulin-naïve patients believe that insulin will not be effective in helping them manage their diabetes more effectively.
4. Disorders that pre-date the onset of diabetes
Conditions such as eating disorders (anorexia, bulimia and obesity), conduct and behavioural disorders, substance-abuse disorders, emotional and psychological difficulties, psychiatric disorders and family dysfunction present additional challenges when insulin is introduced into treatment across all age groups. All of these conditions can lead to resistance in patients. Effective treatment for these disorders will make it easier to establish acceptance of insulin injections but unfortunately, some of these conditions are hard to treat effectively and insulin treatment may still be compromised.

5. Weight loss
Teens are known to stop or reduce dosages of insulin injections and run high blood glucose levels to lose weight. Many have to be hospitalised for diabetic ketoacidosis (DKA). Some become very skilled at having the insulin just before they develop DKA and so can continue insulin omission as a means of keeping their weight down for extended periods.

6. Injections at school or work or in public
Children sometimes feel embarrassed about injecting and testing at school and this may lead to a child avoiding testing and having injections.

Teenagers are sensitive about being ‘different’ and are very conscious of the attitudes of their peers towards diabetes. If their peers react badly and pick on their vulnerabilities, teens with diabetes often avoid injecting or testing at school. One 15-year-old teen was reported by her peers for making them feel sick. Her ‘crime’ was that she performed her finger-prick glucose tests and gave insulin injections discreetly in class before school breaks. The teacher told the girl that that she should go to the bathroom to do the injections thereafter. The need to manage her diabetes made her feel alienated from her peer group. She complied, but her humiliation and isolation into anger against having diabetes. In time, she stopped injecting at school, which had a negative effect on both her diabetes control and her emotional wellbeing.

Adults may also have issues with injections in the workplace or in public. The following examples of patient comments reflect this:
• ‘I have never seen anyone inject in the office. I don’t feel comfortable doing it in public, not so much that they won’t like the sight of the needles, but because I think they will be worried about me contaminating the area with my blood. You know, everyone is scared of AIDS these days.’
• ‘I’m having a problem visualising how I can go to a business meeting, check blood sugar, then inject, in front of everyone. I have no experience with it yet, but I think I would probably go to the restroom before I did that.’
• ‘I’ve never seen anyone inject in public and I would feel better if I did. I do it discreetly at the table in a restaurant because most of the time no one sees or comments. If I do it in the dirty restroom, I usually grossed out and often get looks and comments.’
• ‘I’ve had type 1 for 35 years. When I was younger, I could not inject in public. After about age 40 years I began to loosen up and I am comfortable with it now.’
• ‘One restaurant owner came and asked me to leave because someone had reported that I was shooting up drugs in his restaurant! Now I am reluctant to do it in public.’

7. Treatment burden versus Treatment benefit
Vijan et al (2014) examined a group of patients with type 2 diabetes offered additional treatment, including insulin, for improving their glycaemic control. They found that patients older than 50 years often developed PIR when they perceived the treatment burden to be greater than the benefits. The research found that, in fact, for this age group, the additional treatment offered at most modest benefits. Thus, if the patient is resistant to the added treatment, it may not be beneficial to start it.

8. ‘Insulin Resistance’ in Healthcare Professionals
People with diabetes are sensitive to their healthcare professional’s attitudes and behaviours. These can have a significant impact. Professionals often use insulin therapy as a threat to force people with type 2 diabetes to exercise more and adhere to their ‘diet’ - “If you go on like this, I will have to put you on insulin”. In this context, insulin therapy is seen as a punishment for lack of personal success.

Professionals may speak of ‘failure’ of oral agents, necessitating insulin therapy, when in truth it is just an added treatment requirement as the condition progresses and beta-cell mass declines. Patients interpret the word failure personally - “that really means I have failed”. They develop resultant feelings of guilt and/or anger and become resistant to taking insulin.

When the professionals themselves are reluctant to initiate insulin treatment, even because of true concern for certain patients such as the elderly, those with serious co-morbidities or those with reduced life expectancy, patients may develop a greater fear of insulin than if the professional
portrayed confidence in the insulin therapy as being the treatment of choice. Peyrot et al (2005) found that 50-55% of nurses and general practitioners delay insulin therapy until “absolutely necessary”, but specialists are less likely to do so. Nakar et al (2009) found that in Israel, the main barriers for commencing insulin by physicians included patient compliance (92.3%), hypoglycaemia (79.9%), coping with pain associated with blood tests (53.9%), and pain associated with injections (47.4%).

Unfortunately, some professionals are reluctant to initiate insulin therapy because they feel that type 2 diabetes is not ‘serious’ enough to warrant insulin treatment, or they underestimate the real medical risk of persistent hyperglycaemia. This message can make the patient believe the same thing. Some professionals are understandably fearful of the time needed to start and manage insulin therapy (Polonsky and Jackson 2004).

**Intervention strategies for PIR**

Discover the patients’ understanding and beliefs about insulin and discuss these with them, correcting misconceptions they may have. The mother of a 40-year-old man died from complications of diabetes. Watching her die a painful death, he started to neglect caring for his own type 1 diabetes - he felt there was not much point if this was the outcome. His treatment and self-management had been far superior to his mother’s treatment - once he understood that his life might not end in the same way, he was able to continue his treatment effectively. For patients with type 2 diabetes, it is always a good strategy to inform the patient on diagnosis about the progressive nature of the condition. While oral therapies may work well in the beginning, over time, insulin will be required because of diminishing beta cell mass and sub-optimal insulin production. Then when insulin is needed, the patient is better prepared.

Ensure to manage any underlying psychiatric or emotional disorders or family dysfunctions with psychotherapy or appropriate medications.

Depending on the reason for anxiety, help the patient to work through the feelings first and then assist them to explore solutions. For example if the person has a fear of the pain of injections, discuss or demonstrate ways to handle this. Offer constructive support when the feelings are overwhelming. One 82-year-old woman started insulin in hospital after 30 years on tablets. She stated, “This is the end of the road for me.” She felt she could not go on insulin because she lives in a cottage at her daughter’s home. Everyone else was out at work all day, so if she experienced hypoglycaemia, no one could help her. These valid logistical fears should be resolved, or the insulin, if not really needed in this case, should be avoided. Management of needle phobia starts with correct diagnosis followed by referral for behavioural therapy.

Teachers should handle children who are avoiding insulin at school with discretion until the child is comfortable that other children are not going to victimise or bully him or her because of their diabetes. Even if children are able to take care of their own injections and testing, when they go to school, they need the support of the school staff. Parents must make sure that schoolteachers are given the information and resources they need to support the child’s management during school hours. With the right support, the attitude of the child can change. A well-adjusted ten-year-old child, Vanessa, expressed it by saying, “Insulin is something you need to live, so don’t be afraid to do whatever you need to do. You shouldn’t feel embarrassed doing something to live.”

Once patients are at peace with their diabetes and treatment, they seldom have continued issues with injecting and testing in public. Listen to and try to understand the dilemmas your patient may be experiencing, and support them until they reach this peace.

Restore the patient’s sense of personal control. Suggest, “Try this as an experiment for a month and if you don’t think it has been worthwhile, we can rethink it.” This gives them a choice, and reminds them that they will not lose control of their lives.

Enhance self-efficacy as quickly as possible. Teach the patient to inject using a step-by-step process until they feel comfortable that they can do it independently. This boosts the patient’s confidence. They also realise that the modern needles are very small and fine and that insulin injections are almost painless. Discuss hypoglycaemia, firstly how to prevent it and then how recognise and treat it, should it occur. Explain that it is less common amongst patients with type 2 diabetes.

Spread the good news. Review the positive benefits accrued – improvements in mood, sleep, energy levels and long-term health.

Better diabetes care starts with us as healthcare professionals. We should reflect on, evaluate and challenge our own attitudes, values and beliefs and make changes where necessary.

**REFERENCES AVAILABLE ON REQUEST**
Use of additional technologies in the appraisal of diabetes-related foot disorders - Time to Upgrade

**Introduction**

Uncontrolled diabetes can cause disastrous clinical and lifestyle effects on the feet of patients with the condition and the related financial and social costs are enormous. Sadly, the fact that many people with diabetes and many healthcare professionals do not take diabetes seriously, compounds the problem.

Apart from these obvious challenges, we currently have a number of technologies available to practitioners to assist in the appraisal and management of the feet of a person with diabetes. Recommendations that can be given to patients, including insightful apps, will be also be discussed.

This Journal has previously covered the comprehensive approach to foot assessment in diabetes. It is suffice to say, that only a few resources are required and that establishing a risk profile for each patient, upon which all future management can be based, is paramount. Additional tools and templates are also available to the podiatrist to augment the above assessment.

However, the move from a ‘diabetes foot screen’, to an examination that includes advanced technologies, greatly enhances the ability to diagnose, and more importantly, the ability to predict and prevent complications from occurring. While no perfect ‘crystal ball’ exists, identification of risk and its elimination can and will benefit all who have diabetes, regardless of duration or of the presence of complications. Perhaps these newer technologies will cause practitioners to err on the side of caution, but apart from the risk of incurring additional costs, clinically, this may not be a bad thing.

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A typical foot-screening assessment looks at the skin, the vascular and neurological systems, orthopaedic ranges of motion and the footwear to identify any anomalies that may be causing or promoting ulcers and other complications.

**Skin examination**
Examination of the skin and documentation of the findings is easily undertaken and routine. Skin tone, texture, temperature and colour are typical aspects evaluated. Additionally, is the skin anhydrotic or hyperhydrotic? Are there callus, hard corns, fissures or tinea pedis (in its different guises), or are there any other dermatological anomalies that warrant noting, management or referral.

Dr D Armstrong comments, “A callus on a diabetic patient’s foot is like a breast lump”. Calluses are warning signs and must be taken seriously. However, to remember exactly what a patient’s foot looked like three, six or twelve months ago can be a challenge. The widespread availability of Smartphone cameras with constantly improving image quality allows prompt photo documentation of any finding – this is easy to introduce into the foot examination. The photos can be saved to a computer and the images shared, stored or printed and used for comparative analysis at follow up.

**The Orthopaedic assessment**
Both the obvious architectural derangements as well as the qualitative reduction in joint mobility underlie this assessment. Reduced skin elasticity and diminished joint mobility are thought to be in part due to tissue glycation.

**Circulation - Improving the basic screening**
The fundamental screening tests for circulation disorders are palpation of the pedal pulses and assessment of capillary refill. These provide a good starting point and are skills in which all practitioners should be competent.

Performance of the procedures to calculate the Ankle Brachial Pulse Index (ABI) is a natural extension of palpating the pulses. This simple-to-perform and often overlooked test should become routine. Some clinical podiatric screening templates only demand documenting the presence of pulses by palpation. It is worth noting that the ABI measurement not only provides information about the presence of foot pulses, but also suggests whether the systemic vasculature is compromised or not.

Additional to the above, the use of well-priced, handheld Doppler devices, with their audible component, improves the accuracy of ABI measurement and often the appropriateness of onward referral to a vascular surgeon.

Even if pulses are palpable, pulse characteristics may supply further vital information. Pulses may:
- be monophasic instead of triphasic (one sound at each pulse beat indicating poor arterial wall elasticity / hardening of arteries),
- have a high pitch (indicating stenosis),
- have low tone (indicating reduced flow) or
- be associated with bruits (indicating turbulent flow).

This further information will alert the practitioner to undertake a timeous vascular referral with the intent of reducing poor outcomes. Conversely, not all patients with impalpable pulses will have an abnormal Doppler signal. Doppler device testing may find no circulatory compromise in these patients.

**Improving the Neurological examination**
Well-known tools such as a 10 g Semmes-Weinstein monofilament, 128-Hz tuning fork, TIP THERM®, sharp/blunt objects and the patella hammer provide sound insights into the neurological status of the foot in diabetes and aid in foot ulcer risk-assessment. However, a simple tuning fork can be replaced by a neurothesiometer. This is a device used to screen for peripheral neuropathy by measuring vibration perception threshold (VPT). VPT is also useful in monitoring the progression of an established neuropathy as well as indicating the possibility of future ulcer formation. “A VPT of >25 V was strongly associated with the risk of foot ulceration” (Young et al 1994).

The benefits of TIP THERM® testing can be extended with the use of thermometry. In the neuropathic foot, thermometry is an effective tool in predicting the onset of ulceration. Houghton et al showed that an increase in skin temperature in a neuropathic foot is a sign that pathology is expected. Thus, monitoring the temperature of feet may provide vital predictive information before it is possible to identify other clinical signs of injury.

Consequently, by harnessing the above technologies and incorporating the information gleaned from the neurothesiometer and thermometry into our clinical examination, early prediction of pathologies such as Charcot neuroarthropathy and potential foot ulceration is possible. We can then positively change the course of what may have been an inevitable catastrophe.
The Third Force... What the eye does not see may still be there...

Standard diabetes screening tests ask only for a visual report on skin lesions and callosities. However, even in the absence of visible callosities, increased forces can be present within the foot. These increased forces are invisible to the naked eye of the healthcare practitioner and are unknown to the patient with an insensate foot. The use of ‘force plates’ (versatile, durable piezoresistive / surface resistive force sensors) such as the RsScan, Tekscan or Winpod plates, are additional resources that aid in the clinical examination and foot screening assessment. Force plates measure the vertical forces placed upon them by the foot during gait.

Biomechanically, we try to understand how force translates to impulse (may be defined or calculated as the product of the average force multiplied by the time over which the force is exerted) and how this may be the aetiology or future aetiology of an ulcer or damage to a joint.

A force applied to the foot for a prolonged period, will increase peak plantar pressure. The increased duration of force may cause joint damage or break down the integrity of the skin (low force applied over a long time, analogous to holding a moderate weight for a long time beyond your normal ability to do so).

Similarly, a force applied very rapidly can also increase peak plantar pressure and damage joints or skin integrity (a high force applied over a short time, such as dropping the same weight onto your toe).

Both of these scenarios (a high force over a short period of time [high impulse] or a low force over a longer period of time [a low force, applied for longer, will have the same magnitude of effect]) create an increase in peak plantar pressure. Several studies have shown that increased plantar pressure in a patient with neuropathy is often a prelude to ulceration, or re-ulceration.

Clinically, force plate software allows easy visualisation, quantification, and interpretation of each scenario by graphically depicting the data as force vs. time curves.

The software with most force plates includes an impulse (force multiplied by time) screen that shows the point at which 65 % of force is applied. This shows the exact place of a possible future ulcer and allows us to recommend and prescribe early and correctly placed orthotic intervention, especially in the insensate foot.
We all know that force exists in the X, Y, and Z-axes, but a limitation of the clinically available plates is that they can only measure force on the Z-axis. Thus, they cannot give us a full understanding of all the forces placed on upon the foot in the gait cycle. The inability to measure forces along the X and Y axes means we lose the ability to ‘see’ backwards and forwards, lateral, as well as rotational (combinations of both x and y) shear forces. Video gait analysis is an additional tool that can help us better understand these.

Having said that, understanding and appreciating the compressive force under foot during gait (i.e. the vertical force in the Z-axis), including the duration of the force application, still allows us a better understanding of why an ulcer is there, or of sites with a high future risk of ulceration. Despite their limitations, these plates allow podiatrists to better identify preventable conditions rather than simply manage poor outcomes. They are also useful in managing patients who already have ulcers.

**CadCam orthotics - Improving the accuracy of orthotic design**

Linked to force plates are the relatively new arrival in South Africa of computer aided orthotic design and computer automated milling (CadCam). Linking force plate data with a sound clinical examination further augments the design. This knowledge allows us to prescribe the orthotic with insight into whether we need to remove or redirect force, spread the load, or to support or cushion a segment. When we add a three-dimensional foot scanner to the mix, we can improve the ability to manufacture orthotics in real time. The CadCam and 3D system and force plates are all fully integrated.

The benefits of this system include:

1. A rapid turn-around time and placement of orthotics in shoes. Older systems that use Plaster-of-Paris casts or foam boxes require a few days to a couple of weeks to manufacture before the device is ready for placement in the shoes. CadCam orthotics can be placed within a few hours.
2. Accurate orthotics. Computer aided milling produces a device that is accurate to a tolerance of 0.2 mm. Any subsequent devices will also be identical to the first.
3. The ability to share digital files and seek input from other specialists.

**Tran et al (2012) discuss the fact that patients with type 2 diabetes demonstrate an improvement in HbA1c of 0.5 % when they logged blood glucose and nutritional intake data on their smart phones using various apps**

4. The use of a 3D scan as a very specific, time-based record of a wound.
5. Highly specific placement of cutouts, depressions, lifts and domes.
6. The ability of the software to accommodate various methods of orthotic modelling.

**Smartphone Applications and new technology**

Just about everyone has a Smartphone, and there are applications (‘apps’) for everything. Tran et al (2012) discuss the fact that patients with type 2 diabetes demonstrate an improvement in HbA1c of 0.5 % when they logged blood glucose and nutritional intake data on their smart phones using various apps. Innumerable apps are currently available and the majority of these are designed to assist the patient in keeping track of dietary intakes, blood glucose measurements, medications taken, activity levels as well reminding when next to see the podiatrist or other member of the diabetes team.

**Apps that assist practitioners**

As discussed earlier, the obvious ways in which Smartphone’s and apps can assist management are through the various camera apps. This Author found an app called Mobile Wound Analyser (MOWA). MOWA is an application for advanced management of pressure ulcers, diabetes and vascular disease. It has sophisticated software to analyze images of ulcers according to four parameters (Necrosis, Fibrin, Granulation, and Other). It goes further by giving recommendations on the most appropriate wound dressing. The drawback is that this app was developed in Italy, so the wound care products may not match those available in South Africa. However, the photo documentation ability is in itself a useful tool.

**On the Horizon...**

Eyesense provides glucose readings by using a small photometer implanted in the interstitial fluid beneath the conjunctiva of the eye. It has been shown to perform as well as conventional meters, without the finger prick. Google Wearable Tech, are in the process of developing a soft contact lens that will measure glucose levels in tears and provide a reading every second. That uses a similar tech but does not need to be implanted.
Doctors in Tucson Arizona, headed up by Dr D Armstrong and engineer Dr. Najafi are developing a smart sock that measures foot pressures on a continual basis. Using pressure sensors and optical fibres, ulcer prediction may be as simple as putting on your socks.

Conclusion
Uncontrolled diabetes costs limbs, lives, and treasuries full of cash. The healthcare arena that deals with diabetes needs to complete the move from an era of being reactive, to one of being proactive. The literature on the morbidity, mortality and costs of uncontrolled diabetes is conclusive, with consistent benefit shown from interventional studies aimed at improving care. We now know enough to predict what may transpire, and if we reduce the ulcer rate by 20-50%, it will have a fundamental impact on many people.

This article is important for two main groups, firstly, healthcare providers involved in diabetes care and secondly, healthcare funders

For healthcare providers, the quality of the foot screening should be elevated to a full foot examination that goes beyond merely an audit screening; the quality of the examination can produce vital information that can lead to very positive outcomes. Understanding that some of the equipment is expensive, practitioners can work within groups and share some more expensive equipment like a force plate. However, a Doppler probe should be mandatory.

For healthcare funders, clearly much of the new equipment is expensive. Sadly, in a highly cost-conscious sector, healthcare funders in South Africa are often hostile to the introduction of new codes for technology, until they are convinced that clear clinical benefit is evident. Practitioners need to assist Funders by not duplicating costs and by providing the necessary evidence for these exciting and beneficial technologies. Funders need to be open to the process, and assist healthcare providers with review of the evidence and the provision of coding, which allows fair remuneration for the testing that is available and enables the purchase of this equipment. Ultimately, even though a full foot assessment will initially increase costs, the resultant reductions in the morbidity, mortality and the costs related to the diabetic foot will prove the cost-efficacy of this enhanced care.

If we fail to improve foot risk assessment by using equipment we have at our disposal, when we know we can reduce the morbidity, and mortality associated with the diabetic foot, are we negligent?

REFERENCES AVAILABLE ON REQUEST
Physicians may underestimate the impact of minor hypoglycaemic events associated with insulin treatment in type 2 diabetes
Results from the Global Attitude of Patients and Physicians (GAPP) Online Survey

Insulin is the most effective pharmacotherapy to control blood glucose in patients with diabetes. However, the risk of hypoglycaemia remains an important concern for both prescribing physicians and patients and is one of the most important barriers to optimal glycaemic control.

Severe hypoglycaemic episodes may be associated with confusion and loss of consciousness, and can be fatal.

Patients are unable to manage these episodes alone and require the assistance of another person to administer carbohydrate or glucagon. However, in the event of less severe hypoglycaemic episodes, patients may be able to take corrective actions themselves. Consequently, these events frequently go unreported and are thus unrecognised as being clinically important. Nevertheless, patients may be worried about or fearful of these episodes. As a result, they significantly influence patient functioning and diabetes management. One example of this is that at least one third of patients report maintaining their blood glucose above recommended levels to avoid hypoglycaemia.

The Global Attitude of Patients and Physicians (GAPP) survey was initiated to investigate the prevalence and

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The impact of self-treated hypoglycaemic events. Data were collected using an online questionnaire completed by patients with type 2 diabetes and their physicians in 17 countries in Europe, North and South America, Asia, Africa and Australia. To be enrolled, the patients needed to be 40 years or older and receiving treatment with a long-acting basal insulin alone or in combination with a short-acting bolus insulin as a separate injection. Patients using premixed insulins were excluded.

During the first phase of the study, data were collected from Canada, Germany, Japan, USA and the UK. More than one third of these patients had experienced a self-treated hypoglycaemic event within the last 30 days and a substantial proportion reported that they had missed, mistimed or reduced basal insulin doses in response to the episodes. Nocturnal events were of greater concern than diurnal events.

The second phase of the study analysed data from a further 11 countries, including South Africa. The majority (70 %) of these patients were on a basal-only insulin regimen. The mean duration of diabetes was 8 years, including 4 years of insulin treatment. 28 % of patients reported at least one self-treated hypoglycaemic event in the past 30 days. Overall, the mean number of events reported per patient during that time was 3.8, but events were more frequent in those receiving basal-only insulin compared to those receiving a basal-bolus regimen (4.3 vs. 2.9 episodes, respectively). Approximately one third of the events occurred at night.

Although up to 90 % of patients reported discussing minor hypoglycaemic episodes with their physician, the doctors reported that in the last 30 days, they had discussed minor hypoglycaemia with only approximately half of their patients receiving insulin. Patients being already well educated about minor hypoglycaemia, patients not reporting minor hypoglycaemic episodes and lack of time were the most common reasons given by doctors to explain why they did not discuss this complication. The prescribers estimated that approximately one quarter of their patient’s under-report the frequency or severity of minor hypoglycaemic episodes.

The impact of minor hypoglycaemic events on everyday activities was significant. Almost one-half of the patients said that episodes negatively affected work performance, concentration and physical activities. More than 40 % felt that it took longer than an hour after an episode to regain normal mental functioning. More than two thirds were worried about events during driving, while caring for children, while sleeping and while alone at home, especially when there was limited access to food or drink. Up to one in five patients reported adjusting their insulin in response to a minor hypoglycaemic episode. Self-treated hypoglycaemic episodes were also associated with increased utilisation of healthcare resources, with a considerable number of patients reporting an unplanned visit to their diabetes specialist (26 %), general practitioner (25 %) or hospital emergency department (14 %) consequent to the episode.

In general, the physician respondents underestimated patients’ concerns. The majority (80 %) felt that providing education alone was sufficient to avoid or manage the episodes, whereas approximately one third said they were likely to adjust the insulin dose at least temporarily.

Despite the differences in geography, cultures and economics between the countries included in the first and second phases of GAPP, the results are remarkably similar. In all countries, minor hypoglycaemic events, and particularly nocturnal episodes, are of major concern to patients and have a significant impact on daily living. Attending physicians may underestimate this impact.

The data collected in GAPP indicates that there remains a need for patient education about prevention and management of minor hypoglycaemia and greater clinical surveillance among healthcare providers to identify patients at risk. Furthermore, it may be necessary in some cases to reconsider the prescribed insulin regimen and its interaction with nutritional and activity patterns.

REFERENCES AVAILABLE ON REQUEST
Better connected for better decisions. The Accu-Chek Connect diabetes management system

On 8th August 2014, Roche Diabetes Care announced the launch of the new Accu-Chek Connect diabetes management system, which is designed to streamline diabetes management. The system seamlessly transfers blood glucose data from the Accu-Chek Performa Connect meter to the Accu-Chek Connect mobile app and the Accu-Chek Connect Online web portal - wirelessly and automatically. The Accu-Chek Connect diabetes management system is also designed to help increase patient engagement through an innovative mobile and web-based approach to self-management, and by enabling highly efficient communication between people with diabetes and their healthcare team.

Introducing the Accu-Chek Connect diabetes management system, a comprehensive meter-to-app-to-web system designed to increase patient engagement and office efficiency

People with diabetes who have a better understanding of how their blood glucose results are used to support therapy decisions are more motivated to use their blood glucose monitoring devices insightfully, testing at the right times and frequency, and making the best use of the available data.1,2

The Accu-Chek Connect diabetes management system may help keep patients engaged with their diabetes management by providing tools to help them visualize trends in their blood glucose data so they can make better decisions. Data sharing capabilities enable remote consultations when patients need timely advice or closer monitoring, and the diabetes data can be uploaded from the patients’ mobile device anywhere an internet connection is available, saving valuable time.

The system seamlessly transfers blood glucose data from the Accu-Chek Performa Connect meter to the Accu-Chek Connect mobile app and the Accu-Chek Connect Online web portal - wirelessly and automatically.

Not just another app: the Accu-Chek Connect diabetes management system puts powerful, clinically proven tools in patients’ hands3,5

Proven, structured testing tools support better outcomes
Meaningful and reliable diabetes data is a key component for making the right therapy decisions.3,4 Roche is committed to helping raise awareness of the need for testing at the right times and the right frequency, making each test result count towards better diabetes management.

The Accu-Chek Connect diabetes management mobile app includes a 3-day glycaemic profile tool, a dynamic version of the Accu-Chek 360° View tool. The Accu-Chek 360° View tool is proven to help patients lower their HbA1c when used in collaboration with their healthcare professional.3 The 3-day profile tool in the Accu-Chek Connect app guides the patient through three consecutive days of blood glucose testing at meals and before bed. It provides a snapshot of recent blood glucose results, which can be used to identify patterns.

Structured testing and the Accu-Chek Connect mobile app can be used to help patients and their healthcare team identify patterns in blood glucose levels, identify the causes behind out-of-target blood glucose results, and decide if any adjustments are needed in the patients’ insulin therapy or other areas of diabetes management.

Mobile bolus advisor supports insulin dose decisions
The Accu-Chek Connect mobile app includes the Accu-Chek Bolus Advisor, an insulin bolus calculator activated and configured by the patient’s physician. Using the patient’s current blood glucose, planned food intake and the patient’s insulin therapy settings, the Accu-Chek
Bolus Advisor supports patients with bolus recommendations based on their physician’s guidance. The Accu-Chek Bolus Advisor is clinically proven to result in improved glycaemic control.\footnote{Ziegler R, Cavan DA, Cranston I, et al. Use of an insulin bolus advisor improves glycemic control in multiple daily insulin injection (MDI) therapy patients with suboptimal glycemic control: first results from the ABACUS trial. *Diabetes Care.* 2013; 36: 3613-3619.}

**Seamless data sharing**
The new Accu-Chek Connect diabetes management system is the first web-based tool from Roche designed to make it easy for insulin-using patients, caregivers and healthcare professionals to see useful information that can inspire better health-related decisions and outcomes. The Accu-Chek Connect diabetes management system uses the patient’s mobile device, and the web, to help patients share important diabetes information with their healthcare team.

The Accu-Chek Connect wireless blood glucose meter uses Bluetooth Smart\textsuperscript{®} technology to transfer blood glucose results to the Accu-Chek mobile app. This information then transmits automatically to the Accu-Chek Connect Online web portal where the diabetes data is current, reliable and accessible from any device with an Internet browser.

**Sources:**

**About Roche Diabetes Care**
Roche Diabetes Care is a pioneer in the development of blood glucose monitoring systems and a global leader for diabetes management systems and services. For more than 35 years, the Accu-Chek brand has been dedicated to enabling people with diabetes to live life as normally and actively as possible as well as to empowering healthcare professionals to manage their patients’ condition in an optimal way. Today, the Accu-Chek portfolio offers people with diabetes and healthcare professionals, innovative products and impactful solutions for convenient, efficient and effective diabetes management, spanning from blood glucose monitoring through information management to insulin delivery. The Accu-Chek brand encompasses blood glucose meters, insulin delivery systems, lancing devices, data management systems and education programmes, all of which facilitate both people with diabetes and their healthcare team to achieve improved medical outcomes.

**For more information please contact**
info@accu-chek.co.za or your local Roche representative

*Ref: AHCPI40722*
Currently, we are witnessing an alarming increase in diabetes in South Africa, both in the young and in adults, regardless of background, ethnicity or age.

Many people with diabetes are not aware of the best approach for their diabetes care...

Neither are many of their healthcare providers...

The CDE provides advanced, continuing education, mentorship and accreditation in diabetes care to members of the wider diabetes team.

To find out more about our Healthcare provider training and support, please contact the CDE Provider Network Liaison Department on 011 712-6000 or e-mail Providers@cdecentre.co.za

www.cdecentre.co.za
Presents a Five-Day Advanced Course in Diabetes Care for Health Professionals 2014

DIABETES ~ THE BURDEN, THE RELIEF

Conservative estimates place the prevalence of Diabetes Mellitus in South African adults aged 20-79 years at 6.5% and the prevalence is increasing. 50-85% of persons with the condition are undiagnosed and at risk from disabling and life threatening complications. Diabetes, together with its associated cardiovascular risk factors is one of the leading causes of death, either directly or indirectly, in our population.

Over the past two decades, it has become evident that good control of diabetes, as well as the common co-morbidities of hypertension and the dyslipidaemias, is vital to prevent or delay the devastating long-term complications of diabetes. To achieve this, people with diabetes need to understand their largely silent condition and the correct principles of self-care.

Health professionals often do not have access to updated approaches to a chronic, mostly self-managed condition such as diabetes — vital opportunities for therapeutic interaction and patient education are lost. Additionally, insight is needed into the ever-widening range of available medications and treatment strategies as well as the relationships between cardiovascular and other risk factors and diabetes.

As health services evolve, there is a move towards Team Management of Chronic Conditions. This has resulted in the rest of the Health Care Team (Nurses, Pharmacists, Dieticians, Podiatrists, Biokineticists and others) playing an ever-increasing role in diabetes care.

WHO SHOULD ATTEND THE COURSE?

This is an Advanced Course, and is aimed at Health Care Professionals who have a basic knowledge and understanding of diabetes mellitus. It is designed to give an extensive overview of the core principles of modern team diabetes management, so enabling the participants to understand the condition in sufficient depth, to make a real difference in the lives of people with diabetes. Pre and Post Course multiple-choice knowledge evaluation tests are administered, to allow for evaluation of the learning experience.

Attendance is also part of the contractual requirements for Practitioners wanting to open CDE affiliated “Centre for Diabetes Excellence” Branches.

CPD ACCREDITED

The Course offers 34 contact hours. The Course is accredited to provide 30 CPD points for Medical Practitioners and other Healthcare Practitioners registered with the Health Professions Council of South Africa.

Pre-Course readings will be supplied by e-mail to all delegates and an electronic manual of all speaker notes will be provided on the first day of each Course.

Official completion certificates will be provided to delegates who achieve a mark of at least 60% in the final Post-Course Knowledge Evaluation.

COMMENTS FROM DELEGATES TO PREVIOUS 5-DAY COURSES:

I realise that I had been blundering around in the dark in treating my patients with diabetes and now someone has turned on the light! This a life changing Course. You have reformed my medical practice forever - General Practitioner
It was a superb Course & should result in a marked improvement in the care of people with diabetes - Registered Nurse

I enjoyed the Course thoroughly. I will manage patients with diabetes with more self-confidence. The talks were excellent, well organized and well presented - Registered Dietician

The message that you convey is that you care. The variety of topics was great. The balance between active participation and listening was great. The great teaching skills in all lectures promote learning - Registered Nurse

All speakers were excellent and displayed an impressive knowledge of their subjects. Your commitment as professionals is highly commendable. I learned a lot from this superb Course. Consequently, I will be able to treat my patients better - Medical Specialist

ANSWERS TO FREQUENTLY ASKED QUESTIONS

Presented at: Glenhove Conference Centre, 52 Glenhove Road, Melrose Estate, Johannesburg (Please note change).
Dates & Fees: Available at www.cdecentre.co.za (Click on CDE Education). Early bookings are advised.

No fee increase for 2014!

Course Hours: Five days of lectures, workshops and discussion (08h00 – 17h00).

Dress: Comfortable, smart-casual

Language Medium: English

Course Information: The Course Coordinator
Tel: +27 11 712-6000 / Fax: +27 (0)86 607-9355
E-mail: John@cdecentre.co.za

PROGRAME SUMMARY

The Course is aligned with the latest evidence based treatment guidelines. Case studies and problem solving approaches are a vital part of the learning process.

TOPICS INCLUDE:
• Holistic Team Care Philosophy & Educational Approaches
• Diagnosis, Classification, Pathophysiology & Prevention of Type 1 & Type 2 Diabetes Mellitus
• Other types of diabetes including Gestational Diabetes
• Treatment of Type 1 & Type 2 Diabetes
• Psychological Adjustment to Diabetes
• Meal Planning & Nutrition in Diabetes
• The Importance of Exercise in Diabetes
• The Medical Management of Diabetic Ketoacidosis
• The Foot of the Person with Diabetes
• Acute Complications of Diabetes
• Diabetes as a Micro- & Macro-vascular Disease & Risk Factor Control
• Managed care in diabetes
• Interactive Team-facilitated Case Study Sessions
• Practical Workshop on Self Care Devices & Equipment

OUR MULTIDISCIPLINARY TEAM OF COURSE LECTURERS AND FACILITATORS

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Specialist Physician, Endocrinologist

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Registered Dietician

Gerda Janse van Rensburg
ND Pod SA IIWCC (Toronto, US) PgDipDM (Glamorgan)
Podiatrist

Michael Brown
B. Nursing (Wits) ACDM (Wits)
Diabetes Specialist Nurse

Rosemary Flynn
MSc (Clinical Psychology)
Clinical Psychologist
The FreeStyle Optium Neo Blood Glucose Monitoring System from Abbott Diabetes Care

According to the World Health Organization, the incidence of diabetes is increasing. With a focus on improving patients’ daily diabetes management, Abbott Diabetes Care (ADC) has developed the FreeStyle Optium Neo blood glucose monitoring system, which recently received the European Conformité Européene (CE) Mark.

The system uses an “E Ink screen,” with electronic paper display technology (similar to an eReader). The large, high-contrast, touch screen, designed to be easy to use and easy to see, reads and reflects light like paper, with no glare, even in bright sunlight.

The new, visually informative, icon-driven display provides a sleek design, visual glucose trend indicators, and insulin logging, all delivered in a single device supportive of daily diabetes self-management.

Other benefits include an insulin-dosing feature, which allows healthcare professionals to programme a patient’s insulin regimen into the meter. With the ability to log actual insulin doses by tapping the ‘Up’ and ‘Down’ buttons at the bottom of the display, patients are able to review logged insulin doses and glucose results with their healthcare providers.

“The FreeStyle Optium Neo system is our latest advance based on the innovative technology that is at the heart of our Optium product range. It combines powerful features, ease-of-use, and the ability to analyze important glucose trends in a single device,” said Heather Mason, Senior Vice President, ADC.

“The device’s advanced design and technology enables the tracking of glucose trends and the logging of insulin doses, which, when used appropriately, will assist with patient daily diabetes self-management. This system is one more example of how Abbott Diabetes Care is focused on meeting the needs of patients.”

The Freestyle Optium Neo system will be rolled out across South Africa by October 2014. The system is designed to be used in conjunction with standard FreeStyle Optium blood glucose and ketone test strips, both of which are currently available in South Africa.
The CDE is again offering a series of advanced 1-Day Master Classes for Healthcare Professionals who have a special interest in diabetes. These Courses are designed to provide an in-depth understanding of therapeutic options aligned with the latest principles of diabetes management. Insulin therapy in the management of type 1 and type 2 diabetes and insights into evidence-based treatment guidelines for oral agents are the subject of these cutting-edge sessions.

Healthcare Professionals can look forward to a series of interactive lectures presented by an Academic Faculty of Senior Endocrine and Diabetes Specialists, followed by discussion of relevant case studies.

We will present the following sessions in 2014 (subject to sufficient Registrations to ensure viability):

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<th>Insulin Therapy</th>
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Each Master Class is accredited for CPD points for registered Medical and other Healthcare Practitioners. Please visit the following page on the CDE Website www.cdecentre.co.za/for-healthcare-professionals/diabetes-courses for further information.
Subscribe now to the South African Journal of Diabetes & receive 4 issues!

A journal for the healthcare professional with an interest in diabetes

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